THE SURGEON'S Circular Letter

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A FAR EAST PERIODICAL

OF ARMY MEDICAL SERVICES INFORMATION

MEDICAL SECTION, HQ - FEC, AND UNC

APO 500

Office of the Chief Surgeon
Far East Command

TO: All Medical Service Personnel, Far East Command

On this, the 177th Anniversary of the United States Army Medical Service, it gives me great pleasure to express my appreciation to the members of our service in the Far East Command.

Never before in the long history of our service has there been an equal to the brilliant record you are now maintaining in Korea and Japan. Death-from-wound rates have fallen to an all time low, even surpassing the excellent record made during World War II.

Wounded men in Korea are evacuated rapidly and treated with modern scientific skill, so that the chances of their recovery and return to duty are excellent.

Certainly the heroic deeds in the presence of the enemy and sacrifices of our medical personnel of all ranks in the area of combat have played an important part in obtaining these results.

Without regard for race, rank, nationality or religion of the patient, you have afforded personnel of all United Nations represented here with the highest quality of professional care.

I would particularly like to thank the personnel of all the United Nations providing medical support in Korea. Through your hardships, sacrifices and tireless devotion to duty to accomplish our mission, you are an inspiration to all and reflect the highest tradition of Military Medicine.

To each officer of the Medical Corps, Dental Corps, Nurses Corps, Veterinary Corps, Women's Medical Specialists Corps, Medical Service Corps, enlisted men and attached enlisted women of the Women's Army Corps, working in full cooperation to accomplish our complex task, I express my thanks for a job well done and extend my best wishes for your continued success.

William E. Shambora
WILLIAM E. SHAMBORA
Major General, MC
Chief Surgeon

27 July 1952

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Volume VII - Number 7

JULY 1952

Headquarters
Far East Command
Medical Section
APO 500

ADMINISTRATIVE

ARMY MEDICAL SERVICE OBSERVES 177TH ANNIVERSARY; 27 JULY

In a letter to the Continental Congress dated 21 July 1775, General George Washington, Commander-in-Chief of the Continental Army, requested an organized medical service for his army of 20,000 men maintaining that "the lives and health of both officers and men so much depend on a due regulation of this department." On 27 July 1775, the Continental Congress passed a bill providing for "an Hospital" or Hospital Department, with personnel to include a chief surgeon, one apothecary and 23 subordinate personnel.

The Hospital Department faced many and myriad difficulties. They were called upon to serve in the war of 1812, for example, with a medical provision of one surgeon and two assistants for each of the thirteen additional regiments. In that war, as in the Revolutionary War, surgical practice was at a minimum. Anesthetics were not in use and blood-letting was still considered a cure-all. At that time, "the more blood expended the better the wounds of the viscera, provided life was not extinguished when the hemorrhage had stopped," was the prevailing attitude of surgeons. In both these wars medical service for the sick was a greater problem than the care of the wounded; there were more deaths from disease and wound infection than from enemy action.

From this unpretentious beginning, lacking in the precedents and centralized control of medical facilities and limited by a demand for strict economy, the Hospital Department progressed to the present highly organized and coordinated scheme of military medical service. The early discoveries, the application of practical sanitary measures and the medical practices of the Army Medical Service have not only prolonged the life of its own personnel but that of all people of the civilized world as well.

The first "Surgeon General" was Benjamin Church, MD, and he carried the somewhat awesome title of Director General and Physician-in-Chief of the Continental Army. In the same year of his appointment to the post, Surgeon General Church was replaced by John Morgan, MD, who, counselled by General Washington and with the assistance of his subordinate medical officers, drafted the first hospital regulations. A chapter of these regulations on treatment of the sick gave information which would still be considered as good common sense.

The achievements of the Army Medical Service are many; its rosters are studded with the names of outstanding physicians and pioneers in the field of medicine--Kelser, Beaumont, Waterhouse, Lovell, Vedder, Letterman, Hammond, Reed, Gorgas, Billings, Darnall. Brigadier General Raymond A. Kelser, besides discovering a vaccine for the dread cattle disease, rinderpest, was responsible for determining the mode of transmission of equine encephalomyelitis. Colonel Edward B. Vedder discovered the cause of beriberi, thereby giving a new lease on life to millions whose food consisted principally of rice. Colonel Vedder was also one of the leading research scientists in the field of deficiency diseases. Other outstanding contributors to the growth of Army medicine included an army surgeon, Benjamin Waterhouse, who, in 1800, brought smallpox vaccine to the United States. From it grew the widespread practice of vaccination which has practically removed smallpox from America.

The year 1821 brought William Beaumont, one of the Army's most famous medical officers, to the foreground. Called on to treat a young half-breed who had been accidentally shot in the abdomen, Surgeon Beaumont took the patient to his own home where he nursed him back to health. However, the wound left a fistula which never completely healed, thus enabling Beaumont to observe the process of digestion.

In 1836, Army Surgeon General Joseph Lovell established a collection of medical books that later became known as the Surgeon General's Library. Still later it was changed to the Army Medical Library and finally, this year, it became the Armed Forces Medical Library. It has grown to be the greatest collection of medical reference books in the country.

During the Civil War, Surgeon Jonathan Letterman won prominence by devising the present day system for speedy evacuation of the wounded; in 1862 Surgeon General. William A. Hammond established the Army Medical Museum to house gross material, instruments and other items of significance in medical history. Today the Museum possesses over 5,000 specimens of gross tissue and the largest collection of microscopes in the world. The institution is the only one of its kind in the world open to the public.

Perhaps the Army Medical Service achievement best known to the general public is the discovery by Major Walter Reed and his associates of the mode of transmission of yellow fever in 1900. Major Reed's work was the foundation for Brigadier General William C. Gorgas whose work in preventive medicine made possible the construction of the Panama Canal.

In the same decade, General Sternberg materially advanced the study of bacteriology in this country. He was also responsible for the organization of the Army Dental Corps and the Army Nurse Corps, and the establishment of a surgical hospital at Washington Barracks, forerunner of the present Walter Reed Army Medical Center.

Then there was Surgeon John S. Billings who pioneered in the study of the ill effects of overcrowding, and Major Carl R. Darnall who originated the process of purifying drinking water by liquid chlorination. The application of Major Darnall's work is now world-wide in scope and probably has saved as many lives as any other medical achievement.

Today, Army Medical Service personnel are continuing, each in his own way, to make outstanding contributions to the field of medicine. A compilation and evaluation of these contributions will, undoubtedly, further the art of medical science. Whether in combat or out, they are dedicated to the task of safeguarding and restoring the health of the individual soldier as the best means of "conserving America's fighting strength."

DISTINGUISHED SERVICE CROSS AWARDED POSTHUMOUSLY AMEDS SOLDIER

For extraordinary heroism in action against the enemy in Korea, an officer and three enlisted men recently were awarded the Distinguished Service Cross, posthumously, according to Eighth Army Order. One of the enlisted men was a member of the Army Medical Service.

On September 12, 1951, Private Richard R. Trenholm, a medical aidman with Medical Company, 38th Infantry Regiment, 2d Infantry Division, accompanied a force assaulting an enemy-held hill near Pia-ri. Wounded early in the attack and in great pain, Private Trenholm forced himself to his feet and rendered aid to two other wounded men. While attempting to evacuate the second man for further treatment, he was killed by a burst of enemy fire.

KNOW YOUR CADUCEUS

In the Army of the United States the care and treatment of the sick and the promotion of health generally has for many years been carried on under the symbol or sign of the caduceus.

Investigations indicate that the caduceus had its origin in civilizations much earlier than the Greek and that it symbolized certain vague groups of mystic or magic processes which in the cult of prehistoric men were anterior to medicine, in our sense, but certainly inclusive of it. In the earlier Babalonian figurations the caduceus is not an emblem but a god in itself; in others it is carried in the hand of gods or goddesses as a sign and symbol of supernatural power. This staff in its oldest form was a rod ending in two prongs entwined into a knot.

Apollo, the Sun-god, carried a staff or magic wand which exercised influence over the living and the dead. Apollo, first victor of the Olympic games, was not only a great athlete but was also god of the healing art, "physician and seer," "health giver," "averter of evil," and "physician of the soul."

Apollo gave his wand to Hermes (Mercury) in exchange for a lyre. Hermes, while carrying this wand came upon two serpents knotted together. While fighting, he separated them with his wand. Mythologists explain this as the time the serpents were substituted for the entwined rod. A pair of wings was sometimes attached to the top of the staff in token of the speed of Hermes as a messenger.

Aesculapius, Apollo's son, became the lengendary Greek god of medicine. In the temple erected to him he effected cures and prescribed remedies to the sick in dreams. His daughter, Hygeia, was the goddess of health. While Aesculapius was treating a patient one day a snake entered the tent and entwined about his walking stick, thus conferring upon him the gift of wisdom.

The serpent was sacred to nearly all the gods, both Egyptian and Grecian. It represented immortality or renovation of life and vigor, typified by the periodical change of its skin. In various places the serpent represented knowledge and culture, shrewdness and wisdom, freedom from disease.

The caduceus, with entwined serpents, frequently appeared on the title pages of medical books published in the 16th Century. One of these printers used the caduceus without the wings but a dove hovers overhead and the complete emblem includes a Greek inscription: "Be ye therefore wise as serpents and harmless as doves." This is perhaps the first instance in which the caduceus of Hermes is associated with medicine. The physician to King Henry VIII was the first to employ the caduceus in his crest.

In 1857 Army Regulations directed that the caduceus be part of the army insignia for hospital stewards. In 1902 uniform specifications directed that the caduceus, as we know it, the staff around which are entwined two serpents and at the head of staff two outstretched wings, be worn as a collar ornament of Medical Service personnel. Army Regulations provide that the caduceus be the only article of the uniform peculiar to the Medical Service to symbolize the noncombatant functions of the Service, and of its neutral status on the battlefield under the Convention of Geneva, 1864.

(Adapted from Army Medical Bulletin, No. 32, July 1935).

IMPREGNATION OF CLOTHING WITH MITICIDES AND USE OF INSECT REPELLENTS

The program of prevention of epidemic hemorrhagic fever in Korea by impregnation of clothing with miticides and the use of insect repellents as an individual protective measure have resulted in numerous requests for information on techniques of application of the impregnant and use of the repellent.

Quartermaster issue anti-mite fluid is compounded under various formulations but consists essentially of a mixture of equal portions of dibutylphthallate and benzyl benzoate, plus an emulsifying agent. One gallon of this mixture in 17 gallons of water will make enough solution to impregnate 50 herringbone twill uniforms and pairs of socks.

The procedure for applying the miticide is as follows:

1. Prepare the dipping solution in an oil drum with top removed or other similar container on the basis of one gallon of the miticide per 17 gallons of water. A stick long enough to reach the bottom should be kept in the drum so that the mixture may be kept stirred thoroughly during the dipping operation. A second oil drum or similar container should be placed adjacent to the first drum to receive the excess water wrung from the clothing. Station a man at each drum.

2. Form the men into a line, each carrying his uniform and socks. Each man should stop briefly at the first drum, immerse his clothing long enough for it to be saturated thoroughly with the solution, and then with the assistance of the man stationed at the second drum, wring out the excess solution into the drum. The liquid collecting in the second drum should be returned as it accumulates to the first container.

3. Hang up the clothing to dry, taking care to protect it from rain while it is still wet with the impregnant. As soon as the clothing is dry, it is ready to wear.

Under conditions of field use, a treated uniform will withstand prolonged soaking resulting from rain or wading in fresh or salt water. The impregnant is removed slowly by laundering but still will be effective after two scrubbings with soap and cold water. Hot water laundering removes the miticide more rapidly and should not be used for laundering treated clothing. After the uniforms have been laundered three times in cold water, they should be reimpregnated.

When impregnation of the uniform by total immersion in the miticidal solution cannot be practiced, the individual may secure protection by use of insect repellent. This repellent for individual use is available in two ounce bottles supplied by the QM. The liquid should be applied by hand to the exposed surfaces of the skin, on the face, forehead, neck, behind the ears, ankles, legs, wrists, hands and arms. It should be applied in a similar manner to the edges of the cloth surrounding openings into the clothing such as the bottoms of the sleeves and trousers, the fly, front opening and neck line.

Miticidal fluids can be applied using sprayers of a proper type. As a general rule, impregnation by total immersion is the preferred treatment because inadequate treatment often follows from use of the wrong type of spray equipment. If sprayers are used, they must be of a type that delivers large coarse droplets that will actually wet the cloth and not drift away as mist. A uniform should be covered with at least two ounces of miticide to insure proper protection.

Emulsion treatments are easily applied in QM mobile laundries and such treatment is to be preferred when large amounts of mite proof clothing are to be issued. As mobile laundries utilize centrifugal extractors which remove more liquid from the clothing than would drip out in ordinary line drying, higher concentrations of the miticide are necessary. Clothing should be weighed before treatment and after centrifuging to determine the amount of liquid retained. The concentration of the emulsion should be adjusted so that each uniform takes up from 2 to 2.5 ounces of the miticide. Drying should be accomplished at temperatures less than 160 F. to prevent loss of impregnant thru volatilization.

Reference: TB MED 31, dtd 21 Jan 48, Scrub Typhus Fever.

MALARIA THERAPY

Section II, SGO Circular No. 110, dated 3 July 1952, is quoted for information and guidance of all medical officers:

"Studies at the present time indicate that the administration of primaquine in doses of 15 milligrams daily for 14 consecutive days will markedly reduce the relapse rate of Korean Malaria (Vivax Malaria) and there has been no apparent toxicity from primaquine when given in doses of 15 milligrams once daily for 14 days. Two large groups of troops were treated with this regimen while on a full duty status and no toxic effects were manifested. However, when administering primaquine, one must be cognizant of its potential toxicity which might result in hemolytic anemia, bone marrow depression, methemoglobinemia and abdominal pain. Primaquine in higher dosage levels may produce hemolytic anemia in Negroes more readily than in Caucasians but this has not been observed when 15 milligrams daily has been prescribed. The presence of anemia does not preclude the use of primaquine in the treatment of malaria.

"Primaquine is effective in destroying the tissue phases of the malaria parasite but is relatively ineffective against the blood stages of the organism and should therefore be used in combination with chloroquine for the treatment of acute attacks of Vivax Malaria.

"In treating the acute attacks, the following regimen is recommended:

Chloroquine, 3 doses of 0.3 grams (base) during the first 24 hours and then 0.3 grams (base) once daily for 2 days.

Primaquine is given simultaneously in single daily doses of 15 milligrams for 14 consecutive days."

The following additional Army Medical Service personnel have been awarded the Distinguished Service Cross, Silver Star, Legion of Merit, Soldier's Medal, Bronze Star Medal with "V", Bronze Star Medal or Commendation Ribbon for exceptional bravery in face of the enemy and meritorious service during the Korean conflict. These names are taken from the MRU Roster prepared as of 18 April 1952.

DISTINGUISHED SERVICE CROSS

Roton, Franklin D., Pvt

SILVER STAR

Adams, Floyd D., Jr., Pvt
Anderson, Victor I, Cpl
Atencio, Jonn R., PFC
Baker, Jimmie, Sgt
Beckingham, Theodore, Cpl
Blansett, Joe C., Cpl
Brakeman, William E., PFC
Brooks, John R., 1st Lt, MC
Brown, Clarence J., Sgt
Buckley, John M., PFC
Council, John B. Jr., PFC
Cratty, James W., Sgt
Csepp, Jack J., PFC
Davidson, Wilbur L., Cpl
Dixon, Douglas O., PFC
Dobbins, Ernest, Jr., PFC
Dougherty, Eugene G., Cpl
Dowdy, Marvin L., Pvt
Ellis, David M., Cpl
Eshelman, Richard C., Cpl
Ford, Henry, Sgt
Goodman, Harry A., Sgt
Granados, Nicandor, PFC
Greene, John T., PFC
Gudmundson, Hjalmar, 1st Lt, MC
Gundlach, Raymond W., Sgt
Kenyon, Eugene P.. Cpl
Knezevich, William, Sgt
Lawton, Ora V., Sgt
Mahue, Aurius J., Jr., Cpl
May, Leslie E., PFC
McCowan, Ralph, Sgt
Neagle, Paul E., 1st Lt, MSC
Novak, Martin T., PFC
Olsen, David J., PFC
Orrach, George A., Sgt
Packard, Stanley W., Sgt
Palermo, Eugene B., Sgt
Pickerel, Robert A., PFC
Plessis, Joseph H., PFC
Pryne, Harvey E., PFC
Ramirez, Frank, Cpl
Ramos, Ralph, Sgt
Rapine, Lonnis D., Cpl
Rhodes, Edmond A., 2d Lt, MSC
Romano, Florentino, Sgt
Scheffler, Elmer H., Pvt
Seaborn, Larry, PFC
Smith, Ernest C., Pvt
Takanishi, Morito, Cpl
Tidwell, Vernon H., PFC
Vega, Charles B., PFC
Waas, Richard, Cpl
Webster, George D., Capt, MC
Whitt, John, Jr., PFC
Winter, Wilbert L., Cpl
Wright, William L., Cpl

LEGION OF MERIT

Cirlot, Joseph S., Lt Col, MC
Dredge, Thomas E., Lt Col, MC
French, Sanford W., Col, MC
Glove, Richard P., Lt Col, MC
Markowitz, Isidor, Lt Col, MC
Page, Thomas N., Col, MC
Rogers, James T., Lt Col, MC

SOLDIER'S MEDAL

Ackerman, Robert C., PFC
McCowan, Ralph L., Sgt
Ming, Paul E., SFC
Snow, David R., PFC

BRONZE STAR MEDAL with "V"

Allen, Lloyd B., Sgt
Anderson, Frederick, Sgt
Bacon, William L., Cpl
Barker, Cecil H., Jr., Cpl
Bell, Fred, PFC
Bellair, Lloyd, PFC
Bossman, Edward L., Cpl
Briggs, James H., PFC
Brown, James R., 2d Lt, MSC
Brown, Obed N., Cpl
Bryant, Charlie, Sgt
Calvo, Sam, Cpl
Ceccato, Robert, PFC
Coen, George D., PFC
Colantonio, Fred, PFC
Collom, Glenn M., PFC
Conger, Jack D., Cpl
Crawford, Charles R., PFC
Cronkhite, Linniel, Cpl
Cummings, Frank C., PFC
Dormelly, William J., Cpl
Eves, Robert J., PFC
Feigelson, Herschel, Sgt
Fina, David R., PFC
Fisher, Frank T., PFC
Fitzwater, Carl S., PFC
Giebeler, Douglas R., PFC
Glover, Earl H., Cpl
Goodin, William L., Cpl
Gordon, Kenneth V., Cpl
Graves, Lauris D., Capt, MC
Grubinskas, Charles, Cpl
Guy, Edward J., 1st Lt, MC
Hall, Robert R., Cpl
Hille, Earl A., PFC
Hulett, Wilbur J., Pvt
Hulsey, Thomas C., PFC
Jackson, Leon, Cpl
Jeffrey, Tennis H., Sgt
Johnson, Stephen C., Sgt
Johnson, William H., 1st Lt., MC
Johnson, William H., 1st Lt,, MC
King, Robert L., PFC
Kocjancich, Edward., Cpl
Kurucz, Steve J., PFC
Landeros, Manuel, PFC
Landers, Charles, M/Sgt
Lange, Philip F., 1st Lt., MC
Leddy, John R., Cpl
Love, Frederick E., Pvt
Martin, Vell B., PFC
McCord, Robert G., PFC
McDonald, Jerome, Cpl
McGuire, Daniel T., PFC
McRuer, Alexander, Sgt
Mendoz, Pete, Cpl
Miller, Edger M., Capt, DC
Miller, Richard, PFC
Millican, Loman C., PFC
Mills, Gerald W., Sgt
Milton, Trent E., PFC
Morfey, Donald L., PFC
Morris, Carson, L., Cpl
Olivera, David M., Cpl
Patillo, Paul E., PFC
Peters, Edward D., Sgt
Prato, Charles, Sgt
Quiring, Donald R., PFC
Quiroz, Rafael, Jr., Cpl
Rich, Lewis, J., Sgt
Robichaud, Richard, Cpl
Rogers, John D., Cpl
Rogers, Robert E., Capt, MSC
Rosa, Jesus, Sgt
Scherf, Donald D., PFC
Scott, Lowell, PFC
Selph, William W., Cpl
Sloth, Dale N., PFC
Smith, Thomas L., Cpl
Swerdi, Thomas, Cpl
Templeton, Herbert, PFC
Tesler, Irving, PFC
Thomas, Earl L., PFC
Thomas Ira L., Sgt
Thompson, Silas, PFC
Thornton, Jimmie H., Pvt
Torres, Melquiades, Sgt
Tucker, Lowell G., PFC
Velasco, Epifanio, Cpl
Vining, Robert A., Cpl
Walker, Donald D., Cpl
Wellman, Leland J., PFC
Wheaton, Howard H., PFC
Wisniewski, Raymond, PFC
Withee, James F., Cpl

BRONZE STAR MEDAL

Abate, Valentino, J., Sgt
Abbott, William L., Maj., VC
Adair, Victor R., Sgt
Adelman, Robert A., PFC
Allen, Lloyd B., Sgt
Anderson, Samuel R., M/Sgt
Arredondo, Stanley, PFC
Atkins, John, Capt, MSC
Avery, William G., Maj., MC
Barmore, John Lucia, Maj., MC
Barrett, Roy D., Cpl
Bass, John, Capt, MSC
Baumgartner, Loran, Capt., MSC
Baxter, Roland A., Cpl

BRONZE STAR MEDAL (CONTD)

Bazemore, William F., Sgt
Beach, Prince D., Lt Col, MC
Beard, Earl P., Cpl
Beers, Leo E., SFC
Bowman, Charles J., SFC
Bradshaw, O'Donald, Cpl
Britton, George T., Lt Col, MC
Brown, Jesse F., Lt Col, MC
Brown, William G., Sgt
Bryan, Joseph L., SFC
Capasso, Salvatore, Maj., MSC
Cappy, Andrew Louis, Maj., MSC
Cartwright, Thomas, Capt, DC
Chalfant, George A., Capt., MC
Chardack, William M., Maj., MC
Cirlot, Joseph S., Lt Col, MSC
Clawson, Carroll K., Capt, MC
Coe, Charles A., Jr., Maj, MC
Cohen, Stanley, Capt, MSC
Cole, Richard K., Jr., Capt, MSC
Collazo, Arcadio, Sgt
Conde, Richard L., Capt, MC
Crouch, William L., Sgt
Cutler, Morton T., Capt, MSC
Dailey, Dayton H., Cpl
Dailey, John J., Cpl
Daniels, Leon L., SFC
Davis, Ray C., Sgt
Deakins, John A., Capt, MSC
Dekle, Marion, Sgt
Delagarza, Carlos G., 2d Lt, MSC
Demorset, Paul L., Pvt
Dodge, Harry Weeks, Capt, MSC
Dudney, Newton E., Capt, MC
Dunn, Johnnie, Sgt
Ellis, Vernon A., Sgt
Evans, Robert H., Maj, DC
Fort, Daniel W., Cpl
Fraticelli, Eddie, Capt, MSC
Fulner, Adam C., SFC
Gaham, Thomas A., PFC
Galanski, Nathan, M/Sgt
Garis, Kenneth D., 1st Lt, MSC
Gentry, James D., Cpl
Gotham, Joseph W., SFC
Grandfield, Robert, Sgt
Greene, John T., Pvt
Gregg, Robert A., Capt, MSC
Guerin, Irving J., M/Sgt
Gupton, Donald E., Sgt
Gwinn, Frank W., Maj, MC
Hannon, Joseph L., Capt, MC
Harris, Bertie C., Sgt
Hays, Jack M., Capt, MC
Heaton, Stanley B., Cpl
Hepsh, Harold, 1st Lt, MC
Hess, Harry J., Capt, MSC
Hidaka, Harry T., Maj, MC
Hill, James J., Maj, DC
Hotsenpiller, Harry, Maj, MSC
Hunter, George W., II, Col, MSC
Hurrell, William M., SFC
Jenkins, Douglas J., Sgt
Johnson, William H., 1st Lt, MC
Jones, Herbert A., SFC
Keele, John C., Jr., Lt Col, MSC
Kellen, Robert D., Sgt
Kinnely, Charles R., Capt, MSC
Lamp, Clifford, SFC
Leonard, Wesley E., Maj, MC
Limas, Antonio L., Jr., Sgt
Lucas, John C., Jr., Capt, MC
Maciel, Joseph J., Jr., M/Sgt
Mackey, Don, PFC
Magri, Joseph E., SFC
Makely, George P., M/Sgt
Marlette, Robert H., Maj, DC
Marshall, Linn F., Capt, MSC
Martin, Charles E., Capt, MSC
Matthews, John T., Maj, MSC
Mauk, Ralph B., M/Sgt
May, Frank L., 1st Lt, MC
Mazzola, Russell E., Cpl
McGonigle, John F., Capt, MC
McLean, Donald T., Sgt
Meltzer, Daniel, Capt, MSC
Merrihew, Donald R., Capt, MC
Messenger, Andrew L., Capt, MC
Miller, Charles H., Cpl
Moraitis, Constanti, 1st Lt, MC
Moran, Kenneth J., SFC
Morgan, Nathan T., Sgt
Morrell, Nathan E., 1st Lt, MSC
Morrison, Cecil E., Sgt
Mulford, Todd M., Lt Col, MC
Nebe, Frederick M., Lt Col, MC
Nemeth, Ernest A., 1st Lt, MSC
Nocella, Pearl P., Capt, ANC
Norem, Leroy K., Capt, MSC
Norris, John H., Sgt
Olsen, Earl M., Capt, MSC
Omori, Harry I., Capt, DC
Patten, John C., Capt, MC
Peck, Willard L., M/Sgt
Pitnu, Anthony J., Sgt
Plotkin, Alexander, PFC
Potter, Delbert, PFC
Potter, George H., Sgt
Ramsey, Foster G., Maj, MSC
Riley, John W., Maj, MC
Riley, Winifred G., Capt, WMSC
Roberson, Wonsham B., Capt, MSC
Roberts, Herbert G., SFC
Roberts, William W., 2d Lt, MSC
Robinson, Henry B., Cpl
Rodriguez, Jose M., Maj, MC
Rogers, James T., Lt Col, MC
Ross, Richard H., Lt Col, MC
Russell, Charles A., PFC
Ruth, Charles J., Maj, MC
Saenz, Raymond, SFC
Schaap, Howard C., PFC
Schneider, John A., M/Sgt
Scott, Claude A., Maj, MC
Scott, Russel Jr., 1st Lt, MSC
Shoe, Charles H., Pvt
Siems, Florence, Capt, ANC
Sims, Merle M., Sgt
Skelly, Charles D., Sgt
Smith, Deward E., Sgt
Smith, George A., SFC
Smith, Harold E., SFC
Smith, Louis T., Maj, DC
Smith, Orne D., Lt Col, MSC
Smock, Coy E., SFC
Smull, Helen K., Capt, ANC
Snodgross, D. C., Cpl
Sondag, Roger H., Capt, DC
Sonnier, William Jr., Capt, MC
Southerland, Fred W., 1st Lt, MC
Souza, Ronald D., Cpl
Spainhour, Russell, Cpl
Stewart, Price C., Maj, MSC
Stinson, Robert L., Capt, MSC
Stone, Sidney M., Jr., M/Sgt
Stouffer, Jay E., Capt, MSC
Stout, John, Sgt
Stovall, Sidney L., Lt Col, MC
Stropes, Lloyd R., Lt Col, MC
Taylor, Melvin, SFC
Tempie, Merlin F., M/Sgt
Thomason, Eugene A., Capt, MC
Titus, Elbert D., SFC
Trenholm, Richard R., Pvt
Tucek, Arthur R., Capt, MSC
Turner, Robert E., M/Sgt
Vanderwende, Cornel, Sgt
Walter, Florence A., Capt, ANC
Weidenkopf, Stanley, Lt Col, MSC
Weisman, Eugene J., Capt, DC
Wells, Charles H., Capt, MC
White, James, PFC
Wilkins, Truman, Sgt
Windsor, Donald F., Sgt
Wittlif, Charles L., Capt, MSC
Wolf, Donald C., Sgt
Young, John T., Maj, MSC
Zola, Seymour P., Capt, MC

COMMENDATION RIBBON

Addington, Robert E., Cpl
Anderson, Donald D.A., Cpl
Anidon, Donald A., Capt, MSC
Anselmi, Donald, Sgt
Augustus, Charles A., Capt, MSC
Aune, Gerald O., Sgt
Bannister, Norval L., SFC
Beasley, Cecil M., Cpl
Bentley, Alfred H., M/Sgt
Berman, Joseph, Capt, MSC
Blakely, Durward L., Maj, MSC
Blount, Alvin V., Jr., Capt, MC
Boardman, Vernon R., Sgt
Bonczyk, Henry J., SFC
Brown, Gordon T., SFC
Burns, George F., Cpl
Cappelletti, John D., SFC
Casciano, Anthony A., Sgt
Casetta, Gerald Lee, Cpl
Castiglia, Jerry, Cpl
Cobb, Jack N., 1st Lt., MSC
Coe, Charles A., Maj, MSC
Cooper, Charles E., Sgt
Cotrell, Gerald F., Sgt
Cruikshank, Paul H., Cpl
Daab, Vernon J., PFC
Daughton, Jack E., Sgt
Debruhl, Max H., Cpl
Decker, Evelyn, 1st Lt, ANC
Dedionisio, Carl L., SFC
Dier, Norbert, Sgt
Elliott, Charles E., Sgt
Ellictt, Pete, Sgt
Erwin, William D., Sgt
Evans, George D., Jr., Sgt
Evans, Robert H., Maj, DC
Farina, Ernest, SFC
Faught, William V., Cpl
Faulkner, George, Jr., Sgt
Fields, James S., Capt, MSC
Fisher, Alfred S., SFC
Fitzpatrick, Paul, Cpl
Fleming, John I., M/Sgt
Frada, Louis G., M/Sgt
Freeling, Raymond L., SFC
Futsler, Robert L., Sgt
Ganong, William F., 1st Lt., MC
Gasbarro, Sam, M/Sgt
Geiger, Kenneth G., Cpl
Gillenwater, Kent L., Maj, MSC
Glasscock, Thomas T., Maj, MC
Gray, William, Sgt
Griffin, John J., 1st Lt., MSC

(To be cont'd next issue)

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AR 140-300, 5 May 52: Organized Reserve Corps - Order into the Active Military Service of the United States
AR 210-22, 6 Jun 52: Installations - Emergency Expansion Planning
AR 40-630, 12 Jun 52: Medical Service - Subsistence in Hospital Messes
AR 40-105, C-1, 26 Jun 52:) Medical Service - Stand-
AR 40-105, C-2, 10 Jul 52:) ards of Physical Examination for Commissions or Warrant in Regular Army, National Guard of the United States, Army of the United States, and Organized Reserves
AR 40-635, C-1, 10 Jul 52: Medical Service - Medical Services Account
AR 600-450, C-4, 10 Jul 52: Personnel - Separation for Physical Disability

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SR 31-360-50, 12 Jun 52: Subsistence Supply - Commissaries - Reimbursable Issues to Army Medical Treatment Facilities
SR 40-630-1, 12 Jun 52: Medical Service - Subsistence in Hospital Messes
SR 40-635-2, C-1, 10 Jul 52: Medical Services - Accounting for Medical Services
SR 600-450-5, C-2, 10 Jul 52: Personnel - Evaluation and Separation for Physical Disability

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TECHNICAL

HYPERINSULINISM

G. R. Brosius, 1st Lt, MC, Medical Service, Osaka Army Hospital

HYPERINSULINISM might be defined as the state produced by excessive amounts of insulin in the body whether the insulin is exogenous or endogenous in origin. Spontaneous hypoglycemia denotes a depression of the blood sugar to abnormally low level which occurs without the administration of exogenous insulin.

Seale Harris was the first to call attention to the fact that spontaneous hypoglycemia might be recognized by symptoms resembling those which result from excessive doses of insulin. In 1924 he wrote, "When I saw the insulin reaction of diabetic patients, I realized I had seen many patients not taking insulin who had complained of the same symptoms," i.e., hunger, weakness and anxiety neurosis. On the basis of symptomatology correlated with blood sugar determination, Harris established the presence of spontaneous hypoglycemia in a number of patients. In 1927

Wilder first showed the disorder that could result from hyperinsulinism. He demonstrated the presence of large quantities of insulin in the liver metastases of a patient with carcinoma of the islands of Langerhans.

Hypoglycemia associated with anatomical lesion:

A. Hyperinsulinism

1. Pancreatic islet cell adenoma
2. Pancreatic islet cell carcinoma
3. Diffuse hypertrophy of pancreatic tissue

B. Hepatic disease

1. Toxic hepatitis
2. Fatty liver
3. Diffuse carcinomatosis
4. Ascending cholangitis
5. Von Gierke's disease

C. Hypopituitarism

1. Destructive lesions as chromophobe adenoma
2. Atrophy or infection

D. Adrenal cortical insufficiency

1. Tuberculous granuloma
2. Primary atrophy
3. Destructive neoplasm
4. Amyloid disease

E. Hypothyroidism

F. Lesions of the central nervous system (thalamic)

Hypoglycemia without demonstrable origin

A. Increased secretion of insulin by normal islet cells due to autonomic inbalance

B. Decreased secretion of anterior pituitary or adrenal cortical steroids

C. Excessive oxidation of carbohydrate in severe muscular work

D. Pregnancy or lactation

E. Idiopathic

Clinical Picture:

The pattern of the clinical picture is extremely varied, but in one person the symptoms tend to recur, though they may vary in severity. Attacks usually occur before breakfast or several hours after any meal and are frequently precipitated by physical exertion. Attacks are apt to be more frequent and severe during menstruation. They may last but a few minutes and terminate spontaneously or after the ingestion of food. On the other hand, they may be severe and last for hours and even days and prove resistant to treatment. Occasionally an attack ends fatally. The blood sugar at which symptoms appear varies, some patients are asymptomatic until levels below 40 mg percent and others present classical symptoms with blood sugar from 50-60 mg percent.

The symptomatology is closely related to the nervous system. In fact, the subjective and objective signs of this disease are almost entirely attributable to the effects of hypoglycemia on the central and sympathetic nervous systems. In early and mild attacks, disturbances of the sympathetic nervous system usually predominate. These include sweating, flushing, pallor, numbness of the circumoral region, nausea, chilliness, hunger, epigastric pain, trembling, dizziness, weakness, elevated blood pressure, palpitation and syncope (some people feel these symptoms may be due entirely to the secondary discharge of adrenalin).

Evidence of disturbance of the central nervous system is usually seen in more severe attacks and are attributed to the hypoglycemia per se and are as follows: restlessness, thick speech, diplopia, ocular palsies, episodes suggesting petit mal, positive Babinski, tonic or clonic muscle spasms, convulsions and in extreme cases coma and death.

Psychiatric manifestations may occur in either mild or severe attacks and consist of emotional instability, apprehension, difficulty in concentration, disorientation, amnesia, negativism, mania and unconsciousness. The electro-encephalogram frequently shows focal and widespread dysrhythmia which is usually transient but may persist for days or even weeks.

When the symptomatology is considered, it is easy to understand why these patients often find their way to psychiatrists and neurologists before reaching internists. Kepler recorded the various diagnosis made in 21 cases of hypoglycemia. Among these were hysteria, alcoholic intoxication, acute confusional state, brain tumor, epilepsy and encephalitis. The epigastric pain, fatigue and hunger which is relieved by food may be confused with peptic ulcer.

Diagnosis:

To establish a diagnosis of spontaneous hypoglycemia, it is essential to demonstrate a depression in the blood sugar level. It is of equal importance to establish the cause. Since the periods of hypoglycemia are often transient, various means must be used for their detection. (1) A fasting blood sugar before breakfast is important though it is often normal. (2) If possible, a blood sugar determination should be made at the onset of an attack. If the attack is convulsive in nature, the blood sugar toward the end of the episode may be normal or elevated. (3) The glucose tolerance is of use only in those patients in whom a fasting blood sugar is normal or when it is impossible to get a specimen at the onset of an attack (the curve may be normal, flat or diabetic in form during the first 2-3 hours, but if the blood sugar falls below 50 mg percent, 3, 4, or 5 hours after the stimulating dose of glucose and particularly if it is associated with the development of characteristic symptoms, the diagnosis is established.) (4) When hypoglycemia is not demonstrated by these means, a 24-hour fast should be instituted and blood sugar determination made at 12, 18 and 24 hours.

The relative frequency of the various causes of spontaneous hypoglycemia have been analyzed and according to Conn, 90% of all cases result from one of three causes: (1) Functional hypoglycemia, (2) Hyperinsulinism with demonstrable pancreatic lesion, and (3) Organic disease of the liver.

The typical glucose tolerance curve in hyperinsulinism due to islet cell tumor is characterized by (a) subnormal fasting level, (b) peak rarely exceeds 120 mg percent, and (c) returns to subnormal values within two hours. These values are usually main-

tained throughout the 3rd, 4th and 5th hours. In functional nervous disorders, (a) the fasting blood sugar is usually normal, (b) the one hour specimen is usually slightly higher than the normal post cibal range, and (c) at the end of the 2nd and 3rd hours, hypoglycemia occurs with a tendency to spontaneous return by the 4th or 5th hours. In liver disease, the glucose tolerance curve begins with a subnormal level but rapidly rises to hypoglycemia levels followed by a slow return to normal during the day and subnormal levels after a fast of more than 12 hours.

From the standpoint of management, the chief problem of diagnosis is to differentiate organic from functional hyperinsulinism. A differential diagnosis of the three important causes of spontaneous hypoglycemia should not be difficult if one carefully evaluates the (1) fasting blood sugar, (2) glucose tolerance test, (3) liver function studies, and (4) history and clinical courses.

As already mentioned, in functional hyperinsulinism, the fasting blood sugar is normal. It is abnormally low (50 mg percent or less) in hepatic disease or islet tumor. If it can be demonstrated that the fasting blood sugar is below 50 mg percent, functional hyperinsulinism can be ruled out.

When the glucose tolerance curves of hepatic disease and pancreatic islet tumor are compared, although the fasting of both are low, the remainder of the curves are entirely different. The pancreatic tumor is like the normal curve but is set down to a low level. In the hepatic type, the hypoglycemia fasting sugar is followed by a high plateau diabetic type of curve.

In functional hyperinsulinism, the hypoglycemia attacks come on post prandially. Why should this occur at this time? It is probably an exaggeration of normal response. As the blood sugar rises, it serves as a stimulus to insulin production. The pancreas is extremely sensitive to the normal insulinogenic stimulus and this is probably due to autonomic nervous system inbalance. The blood sugar level falls too far but the adrenal mechanism is efficient and brings it up quickly.

In the case of the islet tumor, they complain of pre-breakfast as well as post prandial attacks, so the history of both makes one think of organic hyperinsulinism. Attacks tend to become worse with time and may be precipitated by a missed meal. What causes the hepatic form to have a diabetic type of glucose tolerance curve? Their ability to oxidize glucose is normal but the part of the blood sugar that should be stored in the liver as glycogen doesn't get in so it stays in the blood giving what looks like a diabetic curve. This type of individual hasn't been storing enough sugar as glycogen so his blood eventually falls. When it gets to the point where the adrenal is stimulated to put out glycogen, there isn't any glycogen available, so the blood sugar keeps going down to hypoglycemic level. These people therefore have a history of pre-breakfast attacks and the only time during the day when they may get attacks is when they miss one or more meals. In the patients with no obvious evidence of liver disease, the function studies are of course helpful.

Deficiency of the adrenal cortex may cause hypoglycemia. The clinical features of Addison's disease should remove the danger of confusing this disease with hyperinsulinism in most cases. Hypoglycemia, though uncommon in this condition, may be the immediate cause of death.

In Simmond's disease due to destruction, atrophy or degeneration of the anterior pituitary, it is not surprising that with the removal of a potent insulin opposing gland, hypoglycemia may occur. This is a relatively unimportant feature of the disease when compared with the symptoms of senile decay. Extreme destruction of the anterior pituitary by tumor growth such as a chromphobe adenoma may result in disturbance of the blood sugar level. Here again the hypoglycemia is but a small part of the symptoms produced by such a tumor.

Hypoglycemia of a mild degree is an uncommon finding in a patient with hypothyroidism. It is rarely of significant severity to cause spontaneous hypoglycemia.

Organic disease of the nervous system - although hyperglycemia is more common when the base of the brain is involved, low blood sugar values are occasionally seen in schizophrenia and subdural hemorrhage.

Exercise, if prolonged as in a marathon race, may cause low blood sugar level. The degree is usually not severe. It is due to rapid depletion of sugar in the circulation and unusual reduction in the glycogen reserves. Lactation, renal glycosuria and severe malnutrition, such as seen in anorexia nervosa, may be complicated by hypoglycemia. In each instance the underlying cause is deprivation of nourishment to the organism.

Treatment falls logically under three headings: (a) the acute attack, (b) conservative measures to prevent attacks, and (c) surgical measures.

The treatment of the acute attack is identical to that used in hypoglycemia induced by overdosage of insulin. Briefly, it consists of any form of glucose if the attack is mild. Should the attack be severe, 0.5 to 1.0 cc Epinephrine may arouse the patient enough to take food by mouth or it may be necessary to give I.V. glucose. Twenty mg of glucose or corn syrup dissolved in 8 oz of warm water given rectally has served in an emergency when sterile solutions are not available.

For the prevention of attacks, diet is effective in bringing relief to many patients with functional hypoglycemia. Conn goes so far as to say that in functional hyperinsulinism, if a trial of this diet for a week does not give relief, the diagnosis is not correct. It is useless in cases of islet cell tumors and should be abandoned in favor of surgery as soon as the diagnosis is made. The quickly absorbable carbohydrate foods are of value in correction of the acute attack but because of the stimulating effect which they have on the secretion of insulin and because of the increased insulin sensitivity which follows their use, they are to be avoided. The carbohydrate content of the diet should not exceed 75 gm and is best provided in slowly absorbable forms such as cereals, bread, vegetables and fruits such as bananas and apples. A liberal protein quota is advocated by Conn (120-140 gm). The glucose derived from protein is slowly released and apparently has no apparent stimulating effect on production of insulin. Fat is allowed in liberal quantities. The diet should be divided into six feedings. Undernourishment is to be avoided as weight loss exaggerates the symptoms. Physical exercise, because of its blood sugar lowering effect, should be restricted.

Surgery is indicated in the treatment when a thorough trial of conservative treatment fails to prevent attacks of hypoglycemia and hepatic, adrenal and pit-

uitary lesions are excluded as the cause. If the diagnosis is believed to be hyperinsulinism due to organic lesion of the islands of Langerhans, surgical intervention should not be delayed since the frequent feedings necessary to ward off attacks often lead to the development of obesity which adds to the difficulties of surgical treatment. Adenomas also tend to undergo malignant changes early so they should be operated upon as soon as the diagnosis is well established.

Spontaneous hypoglycemia, due to lesions of the pituitary or adrenal, is not significantly benefited by extracts of these glands now generally available, although ACTH in early hypopituitarism, and cortisone in Addison's disease with hypoglycemia, may prove beneficial. Treatment is therefore limited to direct restoration of blood sugar. In cases resulting from disease of biliary tract or liver, treatment should be directed toward correcting the underlying disorder. In liver disease, unlike functional hyperinsulinism, a high carbohydrate diet is more beneficial than a reduction in carbohydrates. A liberal protein intake fortifies against further damage to the liver. It is particularly important to give these patients a meal before going to bed. In the majority of cases, there is nothing we can do about degenerative liver disease. There is one relatively rare occurrence where hepatic hypoglycemia is based on ascending cholangitis. If the gall bladder is the source of infection, its removal may bring about a remarkable return of liver function.

THE DENTAL USE OF ICE CARTRIDGES

Lt Colonel Arnold H. Feldman, DC, Dental Surgeon, Ryukyus Command

CONVENIENT size cylinders of ice for dental purposes can be made from cartridges in which local anesthetic is dispensed. These cylinders may be directed to the desired area by means of a modified, cartridge-type hypodermic syringe.

These syringes may be used as an adjunct in testing the vitality of the pulp and also as an aid in the elimination of pain during the administration of a local anesthetic.

The equipment necessary for the preparation of ice cartridges includes a refrigerator with a freezing compartment, a cartridge-type hypodermic syringe, and some used local anesthetic cartridges.

The syringe is modified by increasing the opening through the hub to slightly less than the inside diameter of an anesthetic cartridge. The rubber stopper at the hub end of the cartridge is discarded. The stopper at the plunger end is retained. After cleansing the tube, it is filled with water and placed upright in a small cup or container and stored in the freezing compartment of the refrigerator.

When an ice cartridge is needed, it is removed from the refrigerator and held in the closed palm of the hand until a film of water forms on the inside surface of the glass tube - usually five to ten seconds. The cartridge is now placed in the modified syringe with the rubber stopper next to the plunger and is directed either to the tooth to be tested or to the area of needle insertion. This technic should not be used when there is a possibility of the syringe or ice water making contact with a hyperemic tooth.

The use of ice cartridges in testing the vitality of the pulps is simple, convenient and expedient. The ice is merely directed on the individual tooth being tested. A definite advantage of this technic is that the tooth need not undergo a sudden shock as may be experienced by some of the other methods. Moreover, the application is confined to the individual tooth.

As an adjunct in the elimination of pain during the administration of a local anesthetic, ice cartridges are of especial use with young patients. The mouth is first rinsed and the area of needle insertion touched with Tincture of Iodine. The cartridge, with ice extruding, is then placed in contact with the tissue at this point and held under slight pressure from the plunger for about one minute. The area is again touched with Tincture of Iodine and the local anesthetic is then administered in the usual manner.

Children are especially appreciative of this technic. Let the child feel the cold of the cartridge. Tell him that you are going to put a popsicle in his mouth and only the tooth is to be put to sleep. Keep up a steady stream of conversation while the ice is being applied and during the injection of the local anesthetic. You can perfect yourself to the extent that the child and the adult, too, undergo a minimum amount of discomfort during the administration of a local anesthetic.

MEDICAL COLLAPSE MEASURES IN THE TREATMENT OF PULMONARY TUBERCULOSIS*

Major C. B. Pramuk, MC, 279th General Hospital, APO 53

ALTHOUGH chemotherapy has moved into the limelight in the control of pulmonary tuberculosis in recent years, and resection has found a definite place in the treatment of this disease, "medical collapse measures" must still be considered as vital adjuncts when the phthisiologist plans his therapeutic attack in any particular case. The much older and classic method, artificial pneumothorax, and the more recent artificial pneumoperitoneum, with or without the addition of temporary phrenic crush, are the most noteworthy methods.

A comparison of the two methods is most interesting. Pneumothorax is usually reserved for those cases of "cooled off," unilateral tuberculosis, with cavitation in the apical, or subapical regions of the lung. On the other hand, pneumoperitoneum can be instituted earlier, and in extensive bilateral disease that is not obviously hopeless. It has proven efficacious in the closure of cavities at all lung levels, although results are generally better in ameliorating lower lobe disease.

Pneumothorax at present has few adherents and the procedure should be reserved for highly selected cases. The contraindications to pneumothorax are more numerous than in pneumoperitoneum, noting, how-

*This paper was presented at the Eighth Army Conference on Chest Disease, 20 February 1952.

ever, that the presence of any "downhill," non-tuberculous condition would be self-evident in the undesirability and futility of the initiation of any collapse measure whatsoever. Far advanced fibroid tuberculous disease falls in the same category. Uncontrollable abdominal hernia and dense peritoneal or pleural adhesions offer the principal barrier to pneumoperitoneum.

Pneumothorax carries the following two groups of complications, the operative and the late. Of the former, pleural shock (of debatable pathogenesis, but a definite entity to be considered), mediastinal emphysema, lung rupture, broncho-pleural fistula, and tension pneumothorax following rupture of an emphysematous bleb, are commonly seen. Of the late complications, the most frequent and troubling are pleural effusion, empyema, and the inexpansile lung.

Pneumoperitoneum, on the other hand, has a much more limited set of complications, tuberculous peritonitis and ascites occurring in approximately one percent of cases under treatment. Pelvic inflammatory disease is questionably more frequent when a patient is under this type of therapy. Nausea and pain cannot be classified as complications, however, they must be mentioned as factors, which, at times, lead one to elect cessation of the treatment. It is the relative lack of complications in pneumoperitoneum that has influenced many to turn their attention to this form of therapy.

Complications common to both are air embolism and subcutaneous emphysema. Air embolism, often fatal, is rare whereas subcutaneous emphysema occurs a great deal more frequently, but is of no particular consequence.

The following advantages of pneumoperitoneum over pneumothorax must be kept in mind:

(a) The mental attitude of a patient is given a substantial "boost" when positive therapy is initiated.

(b) The ease of institution of pneumoperitoneum, and the facility of refills (without the use of novocaine, usually), allays much of the apprehension on the part of the patient.

(c) The treatment is reversible; the same definitely cannot be said for pneumothorax. No difficulty is encountered in restarting if temporarily stopped by either doctor or patient for reasons of their own.

(d) In basal lesions, and cavitation in that area, pneumoperitoneum may be particularly efficacious, especially if a temporary phrenic crush is added. In reference to the latter, it seems best to reserve this additional measure for unilateral pathology (phrenic crush is often permanent, although not so intended), and to those cases particularly resistant to diaphragmatic elevation.

(e) A moderate degree of pleural adhesions may be present, yet in spite of these, pneumoperitoneum often effects satisfactory resolution of lesions. Of course, this does not hold for all cases, or in the treatment of fibroid tuberculosis. The same type of adhesions, however, are invariably a definite "stumbling block" in the acquisition of a satisfactory pneumothorax "pocket."

(f) Earlier use of pneumoperitoneum (possible because of the lack of danger of atelectasis and the previously mentioned complications), enhances drainage which is vital in the resolution of tuberculous disease, and the early closure of cavitation obviates the thick-walled lesion which will later require thoracoplasty or a resection.

Comparison of the two forms of therapy used in 200 cases of each type during the year 1951 revealed the following:

(a) Pneumoperitoneum proved the easier to induce and to follow up. Fluoroscopic control was not as vital a factor, although it still remained a desirable procedure.

(b) Pneumothorax did, however, in approximately 75% of the cases that were successfully induced, reveal a favorable trend. Cavity closure was somewhat more dramatic than in pneumoperitoneum, as a rule. Sputum conversion, on the average, turned from positive to negative in a shorter period of time. It must be borne in mind, however, that of the cases in which both forms of therapy were instituted, the average case treated with pneumoperitoneum was definitely more serious, often being in the far advanced group. This conforms with the indications for each type of therapy outlined earlier in this paper.

(c) Streptomycin was not used in all cases, but about equally in both groups. Therefore comparisons of its influence in this series are not possible except to note its influence was approximately equal in both types of procedure.

(d) Our observations supported, generally, the conclusions drawn by many others in regard to the complications in these collapse measures. In pneumothorax, the complications outnumbered those of pneumoperitoneum approximately five to one.

In view of the fact that this paper favors pneumoperitoneum, it might be interesting to add a brief note on the mechanism of this form of collapse measures. With the introduction of a very minute amount of air (even a few c.c.), into the peritoneal cavity, the pressure turns from a negative to a positive one.

This pressure change transmits its effect to the thorax, altering momentarily the intrapleural pressure. Physiology is not permanently affected in the least. Some degree of selective collapse is thereby obtained, the diseased tissue tending to undergo collapse more readily than the healthy lung tissue. The motion of the diaphragm also is limited, lung volume is reduced (on the average 10-15%), and the lung is put to rest. Following the induction of pneumoperitoneum with approximately 500cc of air, and repeated once or twice a few days apart, maintenance is usually kept with refills averaging 1000 cc weekly.

SUMMARY: The selection of therapeutic collapse procedures in the treatment of pulmonary tuberculosis should be highly individual, pneumoperitoneum being much safer, carrying fewer complications, and extremely efficacious in a wider variety of cases than pneumothorax.

Although patients were followed for a relatively short period, experience with pneumothorax and pneumoperitoneum substantially approximated the conclusions of many groups treating pulmonary tuberculosis in the United States.

Reference: "Artificial Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," John L. Elliott, MD, FCCP, and Emil Blair, MD, Savannah, Georgia, Diseases of the Chest, November, 1951.

PRELIMINARY REPORT ON THE EFFECT OF CORTONE IN THE TREATMENT OF PATIENTS WITH VIRAL HEPATITIS

Lt Colonel Tyron E. Huber, MC, Capt Paul E. Lacy, MC, and Capt Andrew T. Wiley, MC., all of U. S. Army Hospital, 8168th Army Unit, Medical Service

THE TREATMENT of hepatitis, either of the so-called infectious or the serum type, has been largely symptomatic and based upon two principles, bed rest and diet. For the vast majority of patients with hepatitis this therapeutic regime is entirely satisfactory and the patient usually makes an uneventful recovery. However, there remain a number of basic and fundamental scientific questions, for the greater part unanswered, relative to the implementation of these two principles in the treatment of patients with hepatitis. For instance, there are no definite scientific data on the length and beneficial effects of bed rest or how long a convalescence is required. Likewise, we have no adequate scientific data to prove whether the high caloric, high carbohydrate, high vitamin, high protein, and low fat diet, so much in vogue during World War II, is the most advantageous diet to be employed in the treatment of patients with hepatitis. Until these basic fundamental questions are answered, the problem of evaluating any therapeutic regimen in the treatment of hepatitis will continue to be a most difficult one.

For the past three years we have undertaken a study to determine the effectiveness, if any, of the newer drugs in our rapidly expanding armamentarium of therapeutics in the treatment of hepatitis. Although the mortality rate has been recorded by Neefe[1] and others as being from 0.2% to 0.5%, actually a very low mortality rate when compared with other diseases, we are nevertheless relatively helpless to alter a process that usually continues on to death when a patient with hepatitis begins to deteriorate and lapses into hepatic coma. Recourse to intravenous glucose is the generally accepted therapeutic approach in such patients. Obviously this procedure leaves much to be desired clinically and from a therapeutic standpoint may be even deleterious if there has been sufficient damage to the hepatic cells to impede phosphorylation and utilization of the glucose.

In a search for a specific therapeutic agent, Shaffer[2] administered aureomycin in acute viral hepatitis and reported little, if any, effect on the duration of the acute symptoms or persistence of abnormal laboratory findings. Recently there has been a resurgence of interest in the use of aureomycin in several different types of liver disease. György[3] has reported that aureomycin apparently was capable of delaying the onset of massive dietary necrosis of the liver in experimental animals. The mode of action of aureomycin is not clear, but it has been postulated that aureomycin suppresses the growth of intestinal flora, thus preventing the elaboration of bacterial metabolic products which the injured liver is theoretically unable to properly detoxify. Moreover, there has been some evidence that aureomycin possesses a rather narrow spectrum of antiviral activity. The most promising results from aureomycin therapy appear to be in cases in which there is impending hepatic coma.[4] However, the results from aureomycin therapy in the treatment of acute infectious hepatitis have been disappointing.

Buchmann[5] used desoxycorticosterone in cases of infectious hepatitis with evidence of improvement in the protracted forms of that disease.

Thorn[6] gave ACTH to one case of homologous serum hepatitis which caused an immediate improvement in the clinical condition of the patient as well as restoration of the liver function tests to within normal limits.

Other investigators have utilized gamma globulin either as an agent of prevention or attenuation in the treatment of infectious hepatitis. In a recent report Stokes[7] reviewed the results using 0.01 ml. gamma globulin per pound of body weight in three institutional epidemics. A single injection of globulin apparently caused protection in the inoculated groups for as long as nine months despite continued intimate exposure to cases of hepatitis in the control groups. Stokes believed this protection to be the result of a passive-active immunization.

With the announcement by Hench[8] in the spring of 1949 that ACTH and cortisone appeared to be beneficial in the treatment of patients with rheumatoid arthritis and other allied conditions, we were immediately impressed with the effects that ACTH and cortisone apparently produced in appetite and resulting weight gain. Of the 21 patients Hench treated with ACTH and cortisone, 20 had increased appetites. The increase was very marked in 11 patients whose appetites became "tremendous or insatiable," and when one patient was allowed to eat ad lib, he consumed 6,000 calories in one day. One patient gained 14 lbs in 22 days; another patient gained 19 lbs in 32 days; and still another gained 26 lbs in 33 days. These patients experienced two types of weight gain. In one type the nutritional gain was steady, progressive, and proportionate to the increased appetite and did not dimish rapidly when cortisone was discontinued. In the other type the weight gain resulted in retention of fluids, was often not accompanied with increased appetite, and often disappeared shortly after cortisone was discontinued. However, it was not always easy to distinguish between the two types or to estimate how much of the increased weight was nutritional until the use of cortisone was discontinued in each patient.

It has been our clinical impression that as long as the hepatitis patient did not develop nausea, vomiting, or aversion to food, and continued to eat, the eventual outcome was usually favorable. Moreover, of the two basic principles involved in the conventional form of treatment, namely bed rest and adequate diet, the latter has appeared to us to be the most important. If the effects of increased appetite and improved nutrition, which Hench had reported following use of cortisone in patients with rheumatoid arthritis, could be produced in patients with hepatitis, then an agent would be available which might possibly alter those processes usually leading to deterioration and death. Yet, however attractive this thought might appear, we were immediately cognizant of a potential therapeutic paradox. For Hench had observed in 1929 that patients with rheumatoid arthritis who developed jaundice seemed to experience improvement in their state of rheumatoid arthritis. In an attempt to explain this phenomenon, Hench had hypothesized some normally occurring product improperly detoxified by a damaged liver, which caused a beneficial effect on rheumatoid arthritis. If this altered metabolic product was cortisone, then cortisone therapy would probably be ineffective in hepatitis where there is obvious hepatic involvement. However, Hench had also reported in 1931 that pregnancy improved the rheumatoid state, yet there is no constant pathology of the liver in the pregnant state. In spite of these theoretical considerations, the mute fact remained that patients given cortisone developed "tremendous and insatiable appetites" and did gain weight. Therefore, we felt that a short-

term preliminary trial with cortisone was certainly worthy of clinical investigation in patients with viral hepatitis. Merck and Company kindly made available enough Cortone* to treat five patients with viral hepatitis.

In order that this study of viral hepatitis could be undertaken with a homogeneous group of patients, those patients with obvious serum transmitted hepatitis, with Weil's disease, syphilis, malaria, typhoid fever, yellow fever, tuberculosis, "chemical and biological hepatitis," and amebic hepatitis, as well as patients with infectious mononucleosis, were excluded. Eight groups of ten patients each were given one of the following forms of therapy:

Group A - Adrenal cortical extract, 10 cc. daily for 7 days (providing approximately 2 mg. of cortisone daily according to Thorn).

Group B - Adrenal cortical extract, 20 cc. daily for 7 days (providing 4 mg. of cortisone daily according to Thorn).

Group C - Adrenalin, 0.3 cc. 1:1000 dilution, plain, for 7 days with a hope that this drug would provoke stimulation of the adrenal cortex through the pituitary-adrenal axis.

Group D - Progesterone, 10 mg. daily for 7 days.

Group E - Progesterone, 20 mg. daily for 7 days. The progesterone was administered on the supposition that a portion of that drug might be transformed into a corticoid.

Group F - Immune globulin in massive doses, 10 cc. daily for 7 days with a hope that a sufficient titre of neutralizing antibodies would be available to attenuate or modify the course of the disease through neutralization of the virus of infectious hepatitis.

Group G - 1,000 cc. 10% glucose in water I.V. daily for 7 days.

Group H - 4 cc. normal saline intramuscularly for 7 days. This group served as control group.

Group I - Finally, only 5 patients were given 100 mg. of Cortone daily for 7 days.

Detailed laboratory studies were accomplished on each of the patients but will not be reported here inasmuch as those studies do not add significantly to our knowledge of hepatitis as previously documented, except that the urinary 17-ketosteroid excretion was found to be in the lower limits of normal early in the disease and to increase slightly after recovery from hepatitis regardless of type of treatment instituted. It would, indeed, be tempting to interpret this low level of urinary 17-ketosteroid excretion as an indication of lowered adrenal activity as hypothesized by Webster[9]. However, the urinary 17-ketosteroid excretion probably depends on a number of different factors in addition to the total amount of adrenal cortical hormone produced and the total metabolic status of the individual patient. In viral hepatitis the element of starvation may possibly play a more dominant role in the reduction in urinary 17-ketosteroid excretion than is commonly supposed, for Landau[10] has reported a marked decrease in the urinary 17-ketosteroid excretion during acute starvation for as short a period of time as four days.

A diet consisting of 400 grams carbohydrate, 155 grams protein, and 120 grams fat, totaling 3,320 calories daily, was prescribed for all patients in addition to multivitamin tablets and 3 grams of Brewer's yeast daily. The patients were removed from bed rest when each had attained normal appetite, when the liver was no longer tender or enlarged, and when the icterus index and other laboratory tests approached normality. The patients were then allowed one week of full activity about the hospital. The laboratory studies were repeated and if there had been no significant change in the patient's condition or in the laboratory studies, the patient was discharged and returned to full duty. The entire group of patients was placed on one large medical ward of the hospital under the supervision of the same physician, nurse, and enlisted personnel.

It appeared that the group treated with Cortone displayed a more rapid fall of icterus index during the week of treatment and during subsequent weeks than any of the other groups and, that of all the other therapeutic agents employed in this investigation, only the groups treated with progesterone (10 mg. daily and 20 mg. daily) exhibited a significant decrease in icterus index. However, we were aware that at least three sources of error were present in this clinical investigation:

1. All of the groups were not treated simultaneously since the arrival of the Cortone was unfortunately delayed and this group was treated separately at a later date.

2. We intentionally selected the most severe case of hepatitis for administration of Cortone for, having such a limited amount of Cortone available, we could ill afford to expend Cortone on patients with only a mild disease.

3. The number of patients in all of the treatment groups except the Cortone-treated group was 10 and in the Cortone group, 5. We realized that the patients treated in each group represented entirely too few cases for significant statistical analysis.

Therefore, the following clinical investigation was devised in which four groups each of 100 patients would be treated with the following therapeutic agents:

Group A - Progesterone, 10 mg. daily, until clinical recovery resulted.

Group B - Progesterone, 20 mg. daily, until clinical recovery resulted.

Group C - Normal saline, 4 cc intramuscularly daily, until clinical recovery resulted.

Group D - Cortone, 100 mg. daily for the first 7 days, then 50 mg. daily until ambulation was begun, and then 25 mg. daily for 2-5 days.

We were particularly interested in determining if cortisone or progesterone would cause significant

*This material (Cortone Acetate) was supplied through the generosity of Elmer Alpert, M.D., Manager, Clinical Research, Merck and Company, Rahway, New Jersey.

increases in appetite as evidenced by weight gains and daily food intake in ounces. Therefore, we established a system whereby the weight of each tray of each patient was carefully weighed before leaving the ward diet kitchen and upon its return. Thus, the difference represented the amount of food consumed in ounces for each meal. All patients were encouraged to eat as much as they desired, and often as many as three separate servings were consumed at breakfast and lunch, but seldom at the evening meal. All in-between meal consumption of food was likewise recorded and a grand total for each patient was posted in his chart daily. Likewise, each patient was weighed each day at the same time, in the same amount of clothes, on the same scales, by the same ward personnel and recorded in the patient's chart. The same precautions were taken as in the original investigation to insure that a homogeneous group of viral hepatitis patients was studied. They were placed in the respective groups according to their admission to the hospital. Likewise, the same officer, nurse, and enlisted personnel as were present with the original investigation were retained on the same ward during this investigation. The same criteria for ambulation, convalescence, and discharge as in the original investigation were continued.

Since four out of five patients treated with Cortone in the first investigation had displayed a transitory glycosuria, we attempted to exclude by urinalysis, fasting blood sugar determinations, glucose tolerance tests, as well as by family histories, the potential diabetics from the Cortone-treated group. Likewise, by careful histories and physical examinations, those individuals with hepatitis who displayed obvious or potential hypertension, gastric ulcers, and unstable emotional backgrounds were excluded from the group. Finally, a psychiatric evaluation by the staff psychiatrist was obtained on each of the individuals in the Cortone-treated group prior to administration of Cortone.

From the data obtained, it appeared that the Cortone-treated group maintained an average total daily intake at a consistently higher level than the other three groups.

Table 1 records the days of jaundice before admission to the hospital, days in the hospital before treatment was begun, days of treatment, days of illness after treatment was started, and total duration of illness. The Cortone-treated group remained on the average, one extra day prior to initiation of treatment in order that preliminary glucose tolerance tests and psychiatric evaluations could be obtained prior to the initiation of Cortone therapy. Inasmuch as there are only 70 patients in this preliminary report treated with Cortone and 89 patients in the progesterone 10 mg. daily group, 90 patients in the progesterone 20 mg. daily group, as well as 90 patients in the control group, we believe that the groups will probably be comparable, except for the additional day of hospitalization required for the Cortone-treated group, when the goal of 100 patients in each group is reached, at which time all the data assembled will be subjected to critical statistical analysis.

TABLE 1

	Saline (4 cc)	Progesterone (20 mg)	Progesterone (10 mg)	Cortone
Days of illness before admission	9.7	9.95	9.38	10.62
Days of jaundice before admission	4.34	3.90	3.67	4.10
Days in hospital before treatment	3.6	3.14	3.26	4.30
Days of treatment	19.3	19.70	19.20	21.63
Days of illness after treatment started	42.7	44.20	43.21	45.50
Total duration of illness	54.7	55.90	54.93	59.94

Table 2 records the average icterus index and average serum bilirubin of the four treatment groups the day before treatment and at weekly intervals thereafter.

TABLE 2

Icterus Index	-1	+7	+14	+21	+28	+35	+42	+49
Saline (4cc)	45.9	25.10	16.20	11.60	10.10	9.30	8.8	9.6
Progesterone (20 mg)	50.20	28.20	19.10	14.90	12.30	10.63	9.9	9.2
Progesterone (10 mg)	50.20	27.40	19.0	13.99	12.40	10.97	10.4	10.4
Cortone	61.89	23.06	15.74	14.70	11.52	9.97	8.92	8.52
Serum Bilirubin								
Saline (4cc)	7.10	3.46	2.15	1.41	1.13	0.96	0.93	0.98
Progesterone (20 mg)	7.55	4.14	2.57	1.97	1.44	1.15	1.06	1.05
Progesterone (10 mg)	7.63	4.22	2.66	1.83	1.45	1.27	1.23	1.20
Cortone	8.90	3.13	2.05	1.84	1.38	1.10	0.98	0.91

Table 3 records the weight gained during the treatment period and total weight gained during entire hospitalization of the four treatment groups. It is apparent that the Cortone-treated group gained more weight during treatment as well as throughout the entire hospitalization period.

TABLE 3

	Weight Gained During Treatment	Total Weight Gained
Normal saline (4 cc)	5.22	7.49
Progesterone (20 mg)	4.91	7.40
Progesterone (10 mg)	5.29	7.91
Cortone	7.15	9.26

Discussion of Some of the Clinical Aspects of Cortone Therapy in the Treatment of Hepatitis.

The three major effects of Cortone therapy in the treatment of hepatitis are:

There was an earlier and more striking amelioration of the symptoms of hepatitis, such as nausea, abdominal discomfort, malaise and depression after the first few days of Cortone treatment, than would normally be expected in the patients treated with bed rest and an adequate diet.

A rapid return of the appetite to normal or above normal levels occurred. Although an increased appetite is characteristic of the recovery stage of hepatitis, the patient treated with Cortone experiences a tremendously increased appetite much earlier than would be expected under the conventional form of therapy.

A more rapid decrease in clinical jaundice follows the institution of Cortone treatment and the decrease begins earlier than would be anticipated in the normal clinical course of this disease.

The four main side effects we have observed are as follows:

Fluid Retention: It was necessary to reduce the dosage of Cortone to 50 mg. daily at the end of the first week in order to control edema which involves the face and sometimes the ankles.

Withdrawal Symptoms: Colbert[11] has mentioned the recurrence of symptoms following the abrupt withdrawal of ACTH in 5 cases of viral hepatitis. Rifken[12] has likewise reported clinical relapse following sudden discontinuance of corticotropin or cotisone in 4 patients with acute homologous serum hepatitis. We observed this same rebound phenomenon in the original investigation in 5 patients with viral hepatitis that were treated with Cortone. In those cases we observed an actual increase in the icterus index and serum bilirubin as well as the return of clinical symptomatology when Cortone was precipitiously withdrawn. Before the present series of 70 cases of viral hepatitis treated with Cortone was undertaken, we investigated several treatment schedules, including those suggested by Hench. We finally adopted the treatment schedule of reducing the 100 mg. daily dose of Cortone to 50 mg. per day at the end of the first week in order to control edema. The 50 mg. daily dose of Cortone was continued until the time of ambulation, when a further reduction to 25 mg. per day was continued for an additional 2 to 5 days. We have reduced to a minimum these rebound phenomena since the adoption of this treatment schedule for the Cortone-treated group.

Glycosuria: Approximately 50% of the patients treated with Cortone exhibited a transient glycosuria. In the majority of the patients the glycosuria was mild and disappeared when treatment was completed; however, one case displayed hyperglycemia and heavy glocosuria which persisted for four weeks after completion of treatment, but did not require insulin therapy. During this same period one patient in the control group developed a severe case of diabetes requiring large doses of insulin.

Psychiatric Disorders: Several of the Cortone-treated patients complained of nervousness, sleeplessness, and an ill-defined sense of oppression during treatment and in two cases this was sufficiently severe to warrant discontinuing the drug. One case treated with Cortone developed a frank catatonic schizophrenia eight days following completion of treatment. However, during the same period two patients in the control group developed major psychoses.

Summary

It would appear that the Cortone-treated group displayed an earlier and more striking amelioration of symptoms, a more rapid return of appetite and definitely increased weight gain, as well as earlier and more rapid clearing of the jaundiced state, than would normally be expected in patients with hepatitis treated with bed rest and an adequate diet.

However, we do not desire to create the impression that all cases of hepatitis should be treated with Cortone, for as was mentioned previously, the vast majority of patients with hepatitis will respond to the conventional therapeutic regimen. Nevertheless, Cortone would appear to be particularly helpful in the treatment of severe cases of hepatitis or in thosecases of hepatitis that deteriorate under conventional therapy.

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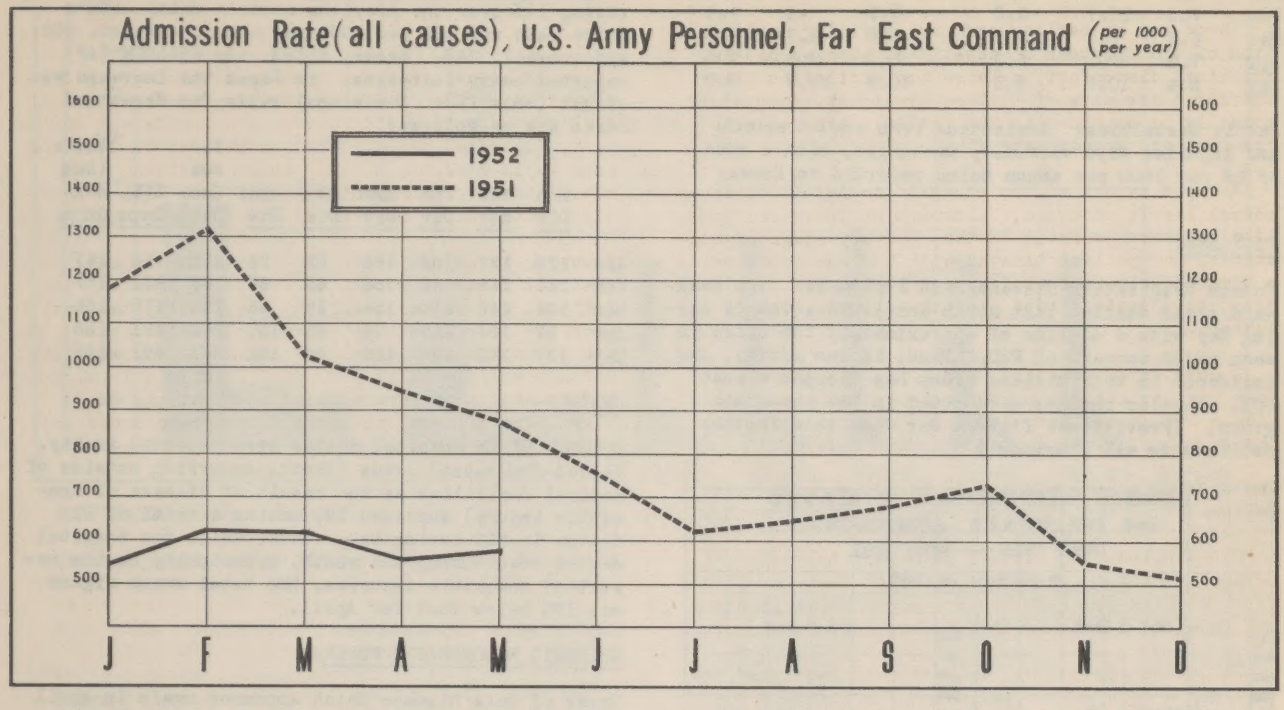
HEALTH OF ARMY TROOPS, FEC



Admissions per 1,000 troops per annum, Army personnel, for the four-week period ending 28 May 1952 were as follows:

	FEC	JAPAN	KOREA	PHILCOM(AF)	RYCOM
All Causes	571	568	587	279	340
Diseases	450	496	439	246	287
Injuries	102	72	119	33	52
Battle Casualties	19	0	29	0	0
Psychiatric	22	14	26	8.2	9
Common Respiratory Diseases and Flu	96	138	79	82	48
Primary Atypical Pneumonia	2.6	1.4	3.3	0	0
Bacillary Dysentery	.18	0	.28	0	0
Amebiasis	.69	1.3	.39	0	1
Malaria, new	13	0	19	8.2	2
Infectious Hepatitis	5.7	5.7	5.3	0	15
Dermatophytosis	6.7	4.2	7.6	0	12
Rheumatic Fever	.40	.12	.56	0	0
Venereal Diseases	246	274	238	205	157
		DAILY NON-EFFECTIVE RATE			
All Causes	17	32	10	30	8.2

Health of the Far East Command for the month of May 1952 refers to Army personnel only.

ADMISSION RATES:

All Causes: The general health of the command remained good during May 1952, with no epidemics reported and no unusual incidence of digestive tract infections. The admission rate to hospital and quarters for "all causes" was relatively unchanged, with a Far East Command rate of 571 per 1000 troop strength. This is 35% fewer admissions than during the same month last year.

Disease: A very slight increase in the disease incidence was noted. Total admission rate for all disease was 450 per 1000 per annum, a figure 30% below last year's rate at this time. Measles, mumps, hepatitis, respiratory diseases, and pneumonia all showed strong declines while hemorrhagic fever, malaria, venereal diseases, and psychiatric conditions increased in incidence. Despite the rise for FEC, the disease rate for Japan was down slightly.

Nonbattle Injuries: Admissions in this category rose 15% during May to a rate of 102 per 1000 per annum. Again, the rate for Japan countered the overall FEC pattern and showed a decline of 4%. The rate for Korea was up by 20%. Approximately one out of every 14 admissions in the nonbattle injury group resulted from automobile accidents and one out of every 10 resulted from athletic injuries. A comparison of the admission rates for Korea and Japan is shown below:

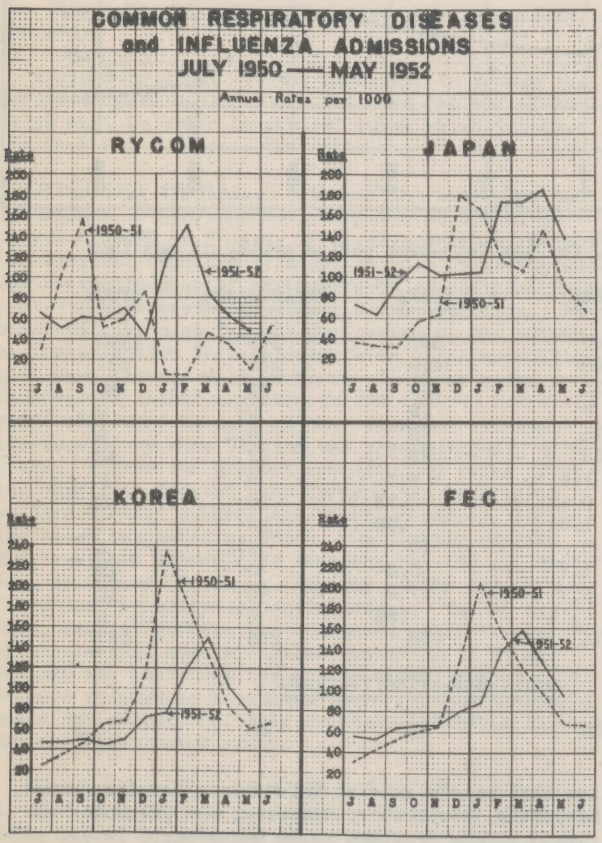
ADMISSION RATES

	Motor Vehicle Accidents			Athletic Injuries		
	FEC	Korea	Japan	FEC	Korea	Japan
Jan	6.9	9.0	2.9	2.3	1.6	3.4
Feb	7.9	10.7	3.3	2.9	.9	6.2
Mar	6.3	8.1	3.7	4.5	2.7	7.1
Apr	7.4	9.1	5.0	6.6	6.5	7.3
May	8.4	10.7	4.2	10.9	11.9	9.8

Battle Casualties: Admissions from combat wounds and injuries were virtually unchanged, with a rate of 29 per 1000 per annum being reported in Korea.

DISEASES:

Common Respiratory Diseases and Influenza: The downward trend started last month continued strongly during May with a decline of approximately 25% noted in each major command of FEC. Thus, in two months, the incidence in this disease group has dropped almost 50%. Similar changes were noted in the pneumonia group. (Provisional figures for June show further declines in all commands.)



Psychiatric: A sharp increase in psychiatric admissions was reported in Korea (24%), while the Japan rate showed a drop of 7%. The Korean rate of 26 per 1000 per annum, while running below last year's rate at this time, represents the highest incidence reported thus far in 1952. (Provisional figures for June show rate increases in both Japan and Korea.)

Malaria: The normal seasonal trend continued during May, reaching a rate of 13 per 1000 per annum, just double last month's rate and virtually the same as the incidence recorded in May 1951. (Provisional June figures show a continued rise, also in keeping with last year's pattern in which the peak was reached in June.)

Venereal Disease: Rates for both white and colored troops increased in the same ratio during May, both rising 18% over the previous month's rate. FEC's white rate was 199 per 1000 per annum; colored, 602; and overall, 246. Korea, RYCOM, and PHILCOM (AF) reported heavy increases; in Japan the increase was slight (only 2%). Divisional rates for Korea and Japan are as follows:

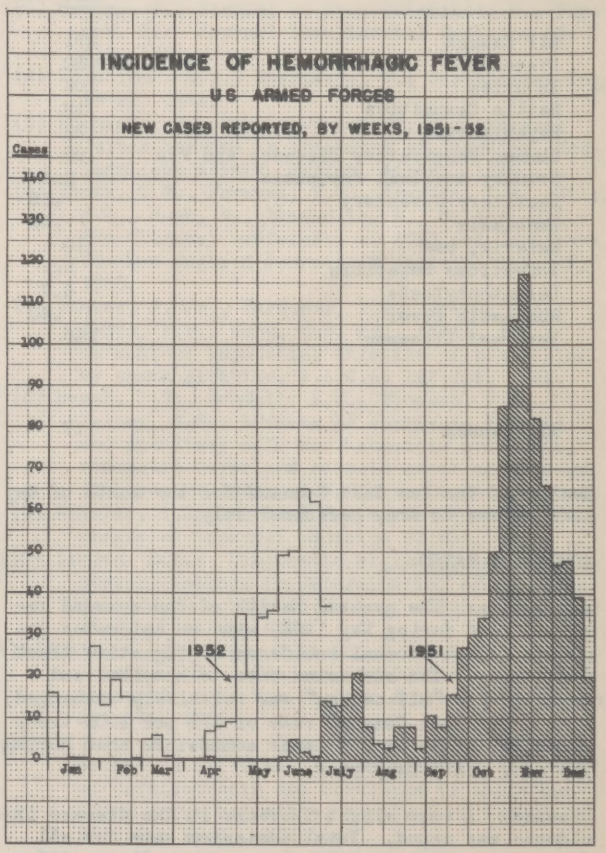
	2nd Div	3rd Div	7th Div	25th Div	40th Div	45th Div	Korea (less Div)	XVI Corps	Japan (less XVI Corps
Jan	159	132	194	183	82	73	273	454	147
Feb	323	189	178	166	66	65	247	431	167
Mar	191	142	210	134	17	48	226	478	166
Apr	97	154	187	74	45	100	260	491	153
May	127	263	176	129	46	132	322	491	145

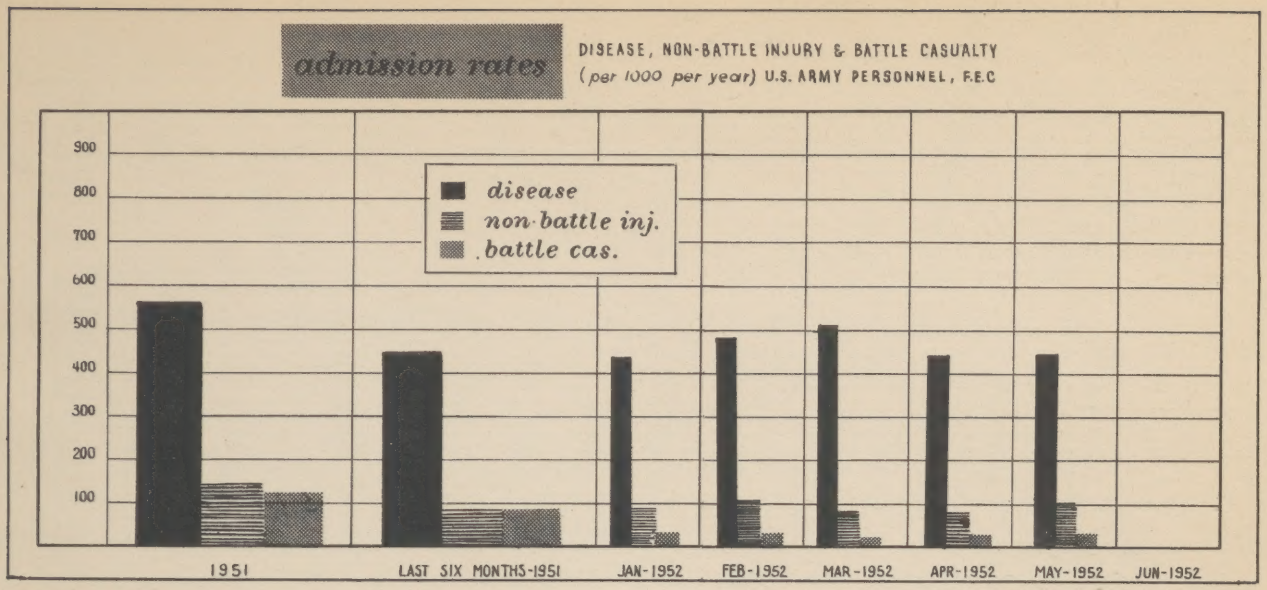
DEATHS:

A total of 52 hospital deaths were recorded in May. Carded-for-record cases (deaths occurring outside of medical facilities as the result of disease or nonbattle injury) numbered 39, making a total of 91 deaths in FEC during May. Thus, while the hospital deaths rose during the month, principally as the result of nonbattle injuries, the total death figure was 13% below that for April.

EPIDEMIC HEMORRHAGIC FEVER:

Cases of this disease which appeared again in April continued to increase during May, as reflected on the following chart:





HOSPITALIZATION: (These data cover all patients, Army, Air Force and others.)

The bed status as of 28 May 1952 was as follows:

	Designated Beds	Operating Beds	Average Beds Occupied: All Patients Army Hospitals	Army Patients: USAF Hospitals	Army Patients: Navy Hospitals
Japan	13,250	10,335	3,543	121	3
Korea	5,400	5,258	2,375	4	56
PHILCOM (AF)	0	0	0	37	0
RYCOM	400	347	194	0	0
FEC	19,050	15,940	6,112	162	59

In Korea, there were 11,700 PsW operating beds, 5,710 of which were occupied by PsW and 1,439 occupied by civilian internees.

The percent of designated beds and operating beds in Army hospitals occupied as of 28 May 1952 was as follows:

	Percent of Designated Beds Occupied	Percent of Operating Beds Occupied
Japan	27	34
Korea	44	45
RYCOM	49	56
FEC	32	38

EVACUATION:

Tabulated below is the number of patients evacuated from the major commands during the four report weeks ending 28 May 1952:

	Evacuated to the Zone of Interior: Army Personnel	Others	Total	Other United Nations Personnel Evacuated to Their Homelands
Japan	509	45	554	45
PHILCOM (AF)	2	0	2	-
RYCOM	25	23	48	-
FEC	536*	68	604	45

*Includes 15 Army patients hospitalized in USAF hospitals.

on the cover

Litter bearers carry wounded soldier of the 23rd Infantry Regiment, 2d Infantry Division, to 2d Battalion forward aid station as battle for possession of "Old Baldy" continues in Korea.

The Chief Surgeon extends an invitation to all Far East Command medical personnel of the U. S. Army, Navy and Air Force, or of the United Nations, to prepare and forward with view to publication, articles of professional or administrative nature. It is assumed that editorial privilege is granted unless author states otherwise.

Capt. Charles A. Copeland, MSC EDITOR